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USPT,PGPB,DWPI	cytochrome c or cyt c	2489	<a href="#">L6</a>
USPT,PGPB,DWPI	(ubiquinone) or (co q) or (coenzyme q)	2037	<a href="#">L5</a>
USPT,PGPB,DWPI	(dye or enzyme or isotope or fluorescent or luminescent )	465553	<a href="#">L4</a>
USPT,PGPB,DWPI	(label or labeled) same (dye or enzyme or isotope or fluorescent or luminescent )	24088	<a href="#">L3</a>
USPT,PGPB,DWPI	(ar nox) or (nox) or (nadh oxidase)	24892	<a href="#">L2</a>
USPT,PGPB,DWPI	((435/4 )!.CCLS. )	2256	<a href="#">L1</a>

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=> s (ar-nox) or (ar nox) or (nadh oxidase)

L1 4194 (AR-NOX) OR (AR NOX) OR (NADH OXIDASE)

=> dup rem l1

PROCESSING IS APPROXIMATELY 29% COMPLETE FOR L1  
PROCESSING IS APPROXIMATELY 63% COMPLETE FOR L1  
PROCESSING IS APPROXIMATELY 93% COMPLETE FOR L1  
PROCESSING COMPLETED FOR L1  
L2 2559 DUP REM L1 (1635 DUPLICATES REMOVED)

=> s ubiquinone or coenzyme q or co q

L3 17262 UBIQUINONE OR COENZYME Q OR CO Q

=> s cytochrome c

L4 78254 CYTOCHROME C

=> s ascorbate

L5 49891 ASCORBATE

=> d his

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FILE 'CAPLUS, USPATFULL, EMBASE, BIOSIS' ENTERED AT 12:56:09 ON 07 AUG 2001

L1 4194 S (AR-NOX) OR (AR NOX) OR (NADH OXIDASE)  
L2 2559 DUP REM L1 (1635 DUPLICATES REMOVED)  
L3 17262 S UBIQUINONE OR COENZYME Q OR CO Q  
L4 78254 S CYTOCHROME C  
L5 49891 S ASCORBATE

=> s l2 and l3 and l4 and l5

L6 9 L2 AND L3 AND L4 AND L5

=> dup rem l6

PROCESSING COMPLETED FOR L6  
L7 9 DUP REM L6 (0 DUPLICATES REMOVED)

=> d ibib abs

L7 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 2000:706975 CAPLUS  
DOCUMENT NUMBER: 133:276372  
TITLE: Methods for identifying agents that inhibit serum  
aging factors (**NADH oxidase**) and  
uses and compositions thereof  
INVENTOR(S): Morre, Dorothy M.; Morre, D. James  
PATENT ASSIGNEE(S): Purdue Research Foundation, USA  
SOURCE: PCT Int. Appl., 43 pp.

CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000057871	A2	20001005	WO 2000-US8433	20000329
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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PRIORITY APPLN. INFO.: US 1999-126894 P 19990330

AB The invention described here relates to methods for prevention or treatment of disorders caused by oxidative damage resulting from generation of reactive oxygen species by an aging-specific isoform of **NADH oxidase (AR-NOX)**. The invention encompasses methods of assaying, screening, and identifying agents that inhibit **AR-NOX**, as well as methods using **ubiquinone** to inhibit the ability of **AR-NOX** to generate reactive oxygen species. These agents may be formulated into pharmaceutical compns. in the prevention and treatment of disorders caused by oxidative damage, such as cancer, diabetes, parkinsonism, atherosclerosis, cardiotoxicity, nephrotoxicity, autoimmune diseases, etc.

=> d 2 ibib abs

L7 ANSWER 2 OF 9 USPATFULL

ACCESSION NUMBER: 1998:150713 USPATFULL  
TITLE: Bioassay for toxic substances activated by metabolic enzyme system  
INVENTOR(S): Read, Harry W., Madison, WI, United States  
Gustavson, Karl, Madison, WI, United States  
Blondin, George A., Madison, WI, United States  
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5843696		19981201
APPLICATION INFO.:	US 1995-551384		19951101 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Knight, John		
ASSISTANT EXAMINER:	Jones, Dameron		
LEGAL REPRESENTATIVE:	Quarles & Brady		
NUMBER OF CLAIMS:	43		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	4 Drawing Figure(s); 4 Drawing Page(s)		
LINE COUNT:	878		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method for bioassaying for metabolic activation of toxicants from a xenobiotic compound by a metabolic enzyme system includes incubating the xenobiotic compound with a metabolic enzyme system known to produce

toxicants during normal metabolic degradation processes and with a mitochondrial membrane preparation competent for enzymatic electron transfer. The production of a toxicant has a detrimental effect upon the electron transfer activity of the mitochondrial membrane preparation which can readily be assayed by observing changes in concentration of a selected redox indicator.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 3 ibib abs

L7 ANSWER 3 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 1997:82388 BIOSIS  
DOCUMENT NUMBER: PREV199799374101  
TITLE: Lipid peroxidation and changes in the **ubiquinone** content and the respiratory chain enzymes of submitochondrial particles.  
AUTHOR(S): Forsmark-Andree, Patrik (1); Lee, C.-P.; Dallner, Gustav; Ernster, Lars  
CORPORATE SOURCE: (1) Dep. Biochem., Arrhenius Lab., Natural Sci., Univ. Stockholm, S-106 91 Stockholm Sweden  
SOURCE: Free Radical Biology & Medicine, (1997) Vol. 22, No. 3, pp. 391-400.  
ISSN: 0891-5849.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB The relationship between lipid peroxidation induced by **ascorbate** and adenosine ADP/Fe-3+, and its effect on the respiratory chain activities of beef heart submitochondrial particles has been investigated.

Lipid peroxidation, measured as thiobarbituric acid reactive substance formation, resulted in an inhibition of the NADH and succinate oxidase activities. Examination of several partial reactions of the respiratory chain revealed inactivation primarily of those involving endogenous **ubiquinone**, i.e., NADH- and succinate-**ubiquinone**, and **cytochrome c** reductases. Ubiquinol-**cytochrome c** reductase, measured with reduced **ubiquinone**-2 as electron donor, was unaffected. The amount of NADH- or succinate-reducible

cytochrome b in the presence of cyanide was strongly decreased, but could be recovered by the addition of antimycin. There occurred a substantial decrease of the **ubiquinone** content in the course of lipid peroxidation, with a linear relationship between this decrease and the NADH and succinate oxidase activities. The results are consistent with

the conclusion that the **ubiquinone** pool undergoes an oxidative modification during lipid peroxidation, to a form that can no longer function as a component of the respiratory chain. Lipid peroxidation also led to a partial inhibition of the succinate dehydrogenase and **cytochrome c** oxidase activities and a minor decrease of the **cytochrome c** and cytochrome a contents. Reduction of endogenous **ubiquinone** prevented lipid peroxidation as well as the concomitant modification of **ubiquinone** and inactivation of the respiratory chain. These observations suggest that the destruction of **ubiquinone** through lipid peroxidation is the primary cause of inactivation of the respiratory chain, and emphasize the antioxidant role of ubiquinol in preventing these effects. The possible implications of these findings for regulation of the cellular turnover of **ubiquinone** by the prevailing oxidative stress are discussed.

=> d 4 ibib abs

L7 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1997:21582 CAPLUS

DOCUMENT NUMBER: 126:102083

TITLE: Lipid peroxidation and changes in the  
**ubiquinone** content and the respiratory chain  
enzymes of submitochondrial particles

AUTHOR(S): Forsmark-Andree, Patrik; Lee, C.-P.; Dallner, Gustav;  
Ernster, Lars

CORPORATE SOURCE: Div. Medical Cell Biology, Karolinska Inst.,  
Huddinge,

S-141 86, Swed.

SOURCE: Free Radical Biol. Med. (1996), Volume Date 1997,  
22(3), 391-400

CODEN: FRBMEH; ISSN: 0891-5849

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The relationship between lipid peroxidn. induced by **ascorbate**  
and adenosine ADP/Fe<sup>3+</sup>, and its effect on the respiratory chain  
activities

of beef heart submitochondrial particles has been investigated. Lipid  
peroxidn., measured as thiobarbituric acid reactive substance formation,  
resulted in an inhibition of the NADH and succinate oxidase activities.  
Examm. of several partial reactions of the respiratory chain revealed  
inactivation primarily of those involving endogenous **ubiquinone**,  
i.e., NADH- and succinate-**ubiquinone** and **cytochrome c**  
reductases. Ubiquinol-**cytochrome c** reductase,  
measured with reduced ubiquinone<sub>2</sub> as electron donor, was unaffected. The  
amt. of NADH- or succinate-reducible cytochrome b in the presence of  
cyanide was strongly decreased, but could be recovered by the addn. of  
antimycin. There occurred a substantial decrease of the  
**ubiquinone** content in the course of lipid peroxidn., with a linear  
relationship between this decrease and the NADH and succinate oxidase  
activities. The results are consistent with the conclusion that the  
**ubiquinone** pool undergoes an oxidative modification during lipid  
peroxidn., to a form that can no longer function as a component of the  
respiratory chain. Lipid peroxidn. also led to a partial inhibition of  
the succinate dehydrogenase and **cytochrome c** oxidase  
activities and a minor decrease of the **cytochrome c**  
and cytochrome a contents. Redn. of endogenous **ubiquinone**  
prevented lipid peroxidn. as well as the concomitant modification of  
**ubiquinone** and inactivation of the respiratory chain. These  
observations suggest that the destruction of **ubiquinone** through  
lipid peroxidn. is the primary cause of inactivation of the respiratory  
chain, and emphasize the antioxidant role of ubiquinol in preventing

these

effects. The possible implications of these findings for regulation of  
the cellular turnover of **ubiquinone** by the prevailing oxidative  
stress are discussed.

=> d 5 ibib abs

L7 ANSWER 5 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1996:321743 BIOSIS

DOCUMENT NUMBER: PREV199699044099

TITLE: Mode of antibacterial action of totarol, a diterpene from  
Podocarpus nagi.

AUTHOR(S): Haraguchi, Hiroyuki (1); Oike, Shingo; Muroi, Hisashi;  
Kubo, Isao

CORPORATE SOURCE: (1) Fac. Eng., Fukuyama Univ., Gakuen-cho, Fukuyama 729

SOURCE: Japan  
Planta Medica, (1996) Vol. 62, No. 2, pp. 122-125.  
ISSN: 0032-0943.

DOCUMENT TYPE: Article

LANGUAGE: English

AB The antimicrobial mechanism of totarol was studied using *Pseudomonas aeruginosa* IFO 3080. This diterpene inhibited oxygen consumption and respiratory-driven proton translocation in whole cells, and oxidation of NADH in membrane preparation. NADH-**cytochrome c** reductase was inhibited by totarol while **cytochrome c** oxidase was not. NADH-DPIP reductase and NADH-CoQ reductase were also inhibited. The site of respiratory inhibition of totarol was thought to be near CoQ in the bacterial electron transport chain.

=> d 6 ibib abs

L7 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1983:402840 CAPLUS

DOCUMENT NUMBER: 99:2840

TITLE: The oxidation of methylated amines by the methylotrophic bacterium *Methylophilus methylotrophus*  
AUTHOR(S): Burton, S. M.; Byrom, D.; Carver, M.; Jones, G. D.

D.;

Jones, C. W.  
CORPORATE SOURCE: Dep. Biochem., Univ. Leicester, Leicester, LE1 7RH, UK

SOURCE: FEMS Microbiol. Lett. (1983), 17(1-2-3), 185-90  
CODEN: FMLED7; ISSN: 0378-1097

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Respiratory activity was studied in *M. methylotrophus* cells grown on trimethylamine with the addn. of dimethylamine, methylamine, methanol, and

**ascorbate**-N,N,N',N'-tetramethyl-p-phenylenediamine. Whole cells of *M. methylotrophus* grown on trimethylamine contained b- and c-type cytochromes, together with cytochromes o and(or) Cco, as the major potential oxidase(s); a3 but not a, was also detected. Such cells exhibited a low rate of endogenous respiration which was dramatically stimulated by the addn. of the other substrates. The anal. of fractions prepd. from *M. methylotrophus* showed that virtually all of the

methylamine dehydrogenase and methanol dehydrogenase activities, together with >1/2 of

the **cytochrome c**, were present in the periplasm, whereas all of the assayable dimethylamine monooxygenase and .apprx.2/3

of the trimethylamine dehydrogenase activities were present in the cytoplasm.

The membranes contained all of the **NADH oxidase** activity and the b-type cytochromes. Apparently, trimethylamine dehydrogenase is assocd. with the cytoplasmic side of the membrane and donates reducing equivs. to the respiratory chain at the level of **ubiquinone**/cytochrome b, whereas methylamine dehydrogenase is loosely attached to the periplasmic side of the membrane and probably interacts with **cytochrome c**; no amicyanin was detected.

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L7 ANSWER 7 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS



ACCESSION NUMBER: 1977:115822 BIOSIS  
DOCUMENT NUMBER: BA63:10686  
TITLE: MEMBRANE BOUND RESPIRATORY CHAIN OF SPIRILLUM-ITERSONII.  
AUTHOR(S): DAILEY H A JR  
SOURCE: J BACTERIOL, (1976) 127 (3), 1286-1291.  
CODEN: JOBAAY. ISSN: 0021-9193.  
FILE SEGMENT: BA; OLD  
LANGUAGE: Unavailable

AB The membrane-bound respiratory system of the gram-negative bacterium *S. itersonii* was investigated. It contains cytochromes b (558), c (550), and o (558) and .beta.-dihydro-NADH and succinate oxidase activities under all

growth conditions. It produces D-lactate and .alpha.-glycerophosphate dehydrogenases when grown with lactate or glycerol as sole C source. Membrane-bound malate dehydrogenase was not detectable under any conditions, although there is high activity of soluble NADH: malate dehydrogenase. When grown with O<sub>2</sub> as the sole terminal electron acceptor, .apprx. 60% of the total b-type cytochrome is present as cytochrome o, whereas only 40% is present as cytochrome o in cells grown with nitrate

in the presence of O<sub>2</sub>. NADH and succinate oxidase are inhibited by azide, cyanide, antimycin A and 2-n-heptyl-4-hydroxyquinoline-N-oxide at low concentrations. The ability of these inhibitors to completely inhibit oxidase activity at low concentrations and their effects upon the aerobic steady-state reduction levels of b- and c-type cytochromes and the aerobic

steady-state reduction levels obtained with NADH, succinate and **ascorbate**-dichlorophenolindophenol suggest the presence of an unbranched respiratory chain in *S. itersonii* with the order **ubiquinone** .fwdarw. b .fwdarw. c .fwdarw. o .fwdarw. O<sub>2</sub>.

=> d 8 ibib abs

L7 ANSWER 8 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 1977:189538 BIOSIS  
DOCUMENT NUMBER: BA64:11902  
TITLE: THE SYSTEMIC FUNGICIDE TRIDEMORPH AS A DUAL SITE INHIBITOR OF THE RESPIRATORY CHAIN OF ELECTRON TRANSFER PARTICLES FROM BEEF HEART MITOCHONDRIA.  
AUTHOR(S): MUELLER W; SCHEWE T  
SOURCE: ACTA BIOL MED GER, (1976) 35 (6), 693-708.  
CODEN: ABMGAJ. ISSN: 0001-5318.  
FILE SEGMENT: BA; OLD  
LANGUAGE: Unavailable

AB Tridemorph (N-tridecyl-2,6-dimethylmorpholine) inhibits both the **NADH-oxidase** and the succinate-**cytochrome c** oxidoreductase system of non-phosphorylating electron transfer particles from beef heart. The concentration required for half-inhibition was 3.4 .mu.M and 24 .mu.M, respectively. Two different sites of action

in the respiratory chain could be localized using difference spectroscopy and

measurements of enzymic activities in various partial systems. The inhibition of the NADH-**ubiquinone** oxidoreductase activity, the suppression of the NADH-induced reduction of all cytochromes and the insensitivity of the NADH-ferricyanide oxydoreductase system indicate a site of action similar to rotenone. The partial suppression of the succinate-induced reduction of cytochrome b with simultaneous complete inhibition of the reduction of the other cytochromes indicate an additional site of action analogous to antimycin A. Both inhibitory actions appeared instantaneously after the addition of tridemorph and

were counteracted by serum albumin. Tridemorph inhibited the oxidation of

external ferrocycytochrome c but not that of **ascorbate**  
/tetra-methyl-p-phenylene-diamine-HCl (TMPID) showing that it is not a  
true inhibitor of the cytochrome oxidase. The TMPD-induced bypass of the  
succinate oxidation was inhibited as well. The possible role of the  
inhibition of the main pathway of the respiratory chain for the  
fungicidal  
action of tridemorph is discussed.

=> d 9 ibib abs

L7 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1972:561840 CAPLUS

DOCUMENT NUMBER: 77:161840

TITLE: Comparison of the **NADH oxidase**  
electron transport systems of two obligately  
chemolithotrophic bacteria

AUTHOR(S): Sadler, Martha H.; Johnson, Emmett J.

CORPORATE SOURCE: Sch. Md., Tulane Univ., New Orleans, La., USA

SOURCE: Biochim. Biophys. Acta (1972), 283(1), 167-79  
CODEN: BBACAQ

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The **NADH oxidase** electron-transport systems of two  
obligate autotrophs were investigated. Cytochromes c547, c550, c552, b  
or

c554, b558 and a were found in *Thiobacillus neapolitanus*. and cytochromes  
c549, 551, c or b555, a1, and a or a3 in *T. thioparus*, A sol. cytochrome  
c552 not present in the particulate fractions was detected in *T.*  
*neapolitanus*. Low potential c-type cytochromes were found in both  
organisms. NADH reduced both cytochromes c547 and c550 in the large  
particle fraction of *T. neapolitanus*, but only c550 in the small particle  
fraction. Both organisms contained the **ubiquinone**, Q-8. The  
levels of flavine, quinone, and **cytochrome c** were  
comparable to those of heterotrophic bacteria. No naphthoquinone was  
detected. The levels of NADH and **ascorbate** oxidases were  
similar to those of heterotrophic bacteria, while NADH dehydrogenase and  
**ascorbate:N, N, N', N'-tetramethyl-p-phenylenediamine.2HCl** oxidase  
levels were higher. In *T. thioparus*, **NADH oxidase**  
activity was located exclusively in the large-particle fraction, and in

*T.*  
*neapolitanus* in both the large- and small-particle fractions. The  
**NADH oxidase** activities of both organisms were sensitive  
to inhibitors usually employed in studies of electron transport.  
**NADH oxidase** of *T. thioparus* was completely inhibited by  
KCN, while that of *T. neapolitanus* was never inhibited by more than 80.  
**Ascorbate** and **ascorbate:TMPD** oxidases were sensitive to  
KCN but insensitive to 2-heptyl-4-hydroxyquinoline N-oxide.  
Electron-transport pathways are proposed for both organisms.

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